Specifically, ginsenoside $Rb_1$ is expected to exhibit efficacy against myocardial infarction, angina pectoris, aortitis syndrome, acute peripheral artery occlusion, thromboangitis obliterans, arteriosclerosis obliterans, Raynaud's disease and Raynaud's syndrome. This is explained in more detail by using the example of myocardial infarction. If a branch of the coronary artery is permanently occluded and is not reperfused, the myocardial cells, to which nutrition is supplied only by the permanently occluded coronary artery branch, enter necrosis.

However, myocardial cells (myocytes) near the necrotic (infarcted) area, such as cells to which blood is supplied, in part, from the other coronary artery branches, can survive as a result of intravenously infused ginsenoside $Rb_1$-mediated upregulation of the cell death (apoptosis)-suppressing gene (Bcl-$X_L$) expression (JP98/365560 and PCT/JP99/02550: "Brain cell or nerve cell-protective agents comprising ginsenoside $Rb_1$"). Subsequently, as a result of ginsenoside $Rb_1$-induced vascular regeneration and reconstruction at the same site, the myocytes can be permanently rescued from cell death. Ginsenoside $Rb_1$ protects patients who suffered from myocardial infarction, but unfortunately underwent no coronary artery bypass or PTCA (percutaneous transluminal coronary angioplasty). It does this through at least two different actions as described above, and is expected to contribute to a reduction in the infarcted lesion. Needless to say, if ginsenoside $Rb_1$ is administered

intravenously to patients, who have developed myocardial infarction before they undergo coronary artery bypass or PTCA, the prognosis of the patients is significantly improved. Furthermore, for patients with diseases of peripheral tissues, the effects and efficacy of ginsenoside $Rb_1$ may be elicited at doses equivalent to doses used for brain diseases, or even at 1/10 to 1/100,000 of those doses.

In this regard, matters which should not be forgotten are that in many cases of patients with myocardial infarction, the pumping function of the heart has deteriorated to cause insufficient cerebral blood flow. This sometimes leads to irreversible brain damage. The intravenous administration of ginsenoside $Rb_1$ contributes to improvement in QOL (Quality of Life) for patients with myocardial infarction, since it protects brain cells or nerve cells against cerebral blood flow failure as described in JP98/365560 and PCT/JP99/02550 ("Brain cell or nerve cell-protective agents comprising ginsenoside $Rb_1$").

Next, the actions of intravenously administered ginsenoside $Rb_1$ are explained in detail.

First, we examined the actions of intravenous infusion of ginsenoside $Rb_1$. For this purpose, for example, male SH-SP rats, weighing 250 - 300 g at the age of 12-13 weeks were used. The animals were bred in an air-conditioned room with a 12:12 hour light-dark cycle, and water and feeds were supplied ad

libitum. The cortical branch of the left middle cerebral artery (MCA) was coagulated and cut under inhalation anesthesia. A single intravenous administration of ginsenoside $Rb_1$ dissolved in physiological saline was conducted immediately after MCA permanent occlusion (6 μg or 60 μg), thereafter continuous intravenous administration of ginsenoside $Rb_1$ was performed for 28 days by using Alza osmotic mini-pump (6 μg/day or 60 μg/day).

Control animals with MCA occlusion (ischemic control animals) and sham-operated animals were administered with the same amount of physiological saline.

After MCA permanent occlusion, according to the method of the inventors of the present invention (Sakanaka and Tanaka)(Igase, K. et al., J. Cereb. Blood Flow Metab., 19, 298-306, 1999; Zhang B. et al., J. Stroke Cerebrovasc. Dis., 7, 1-9, 1998), water maze tests were performed for 4 days at the 2nd week and the 4th week, respectively, and the place navigation abilities of SH-SP rats were determined.

Results are shown in Fig. 1. The left drawing in Fig. 1 shows the results of the 2nd week and the right drawing shows the results of the 4th week after permanent MCA occlusion. In Fig. 1, closed circles (●) indicate the results of rats with sham operation; and open circles (○) indicate the results of MCA-occluded rats administered with only physiological saline; closed squares (■) indicate the results of MCA-occluded rats administered with ginsenoside $Rb_1$ in a dose of 6 μg/day and open